Table 3. Independent predictors of US detected inflammatory findings

	p	Odds ratio	95% C.I.	
			Lower	Upper
ESR (mm/h)	0.039	1.04	1.002	1.078
Time (months) from symptoms onset	0.1	0.924	0.841	1.015

	Total n= 57	US inflammatory findings n= 20	Non-US inflammatory findings n=37	p
Age	55.8 ± 15.2	53.9±14.5	59.5±16.2	0.2
Sex	41 (71.9%)	13 (65%)	28 (75.7%)	0.3
Smoking	27 (47.4%)	10 (55.6%)	17 (50%)	
n= 52	20 (35.1%)	6 (33.6%)	14 (41.2%)	0.8
Smoker	5 (8.8%)	2 (11.1%)	3 (8.8%)	
Former smoker	7 (12.3%)	3 (15%)	4 (10.8%)	
Monocuticular	8 (14%)	5 (25%)	3 (8.1%)	0.9
Extension	42 (73.7%)	12 (60%)	30 (81.1%)	
Polycuticular	11.4 ± 10.4	7.3±6.2	13.5±11.5	0.049
Time (months) from symptoms onset	19.1 ± 21	29.3±28.4	14.5±14.8	0.044
ESR (mm/h)	0.6 ± 1.4	1.1±1.9	0.4±0.9	0.15
CRP (mg/dL) n=47	64.3 ± 321.6	21±53.7	88.5±399.7	0.5
RF (IU/mL) n=53	52.2 ± 231.4	104.8±360.5	22.3±103.6	0.2
CCP (IU/mL)				

Table 1. Baseline characteristics of patients with IA

Conclusion: PD US inflammatory findings are found in 1 over 3 patients with IA being PD synovitis the most common finding, specially at the wrists and MCP joints. Higher ESR values were significantly associated with the presence of US inflammatory findings. Our data highlights how the use of PD US may be useful to detect subclinical synovitis in patients with IA.

	US synovitis	PD positive
Hands	14 (24.6%)	11 (9.3%)
Wrist	14 (24.6%)	11 (9.3%)
MCP	7 (12.3%)	7 (12.3%)
PIP	1 (1.8%)	0 (0%)
Feet	8 (14%)	4 (7%)
Ankle	4 (7%)	3 (5.2%)
Tarsal joints	1 (1.8%)	1 (1.8%)
MTP	3 (5.3%)	0 (0%)
	US tenosynovitis	PD positive
Hands	4 (7%)	3 (5.3%)
2nd, 3rd, 4th or 5th flexor	3 (5.3%)	3 (5.3%)
4th extensor	1 (1.8%)	1 (1.8%)
6th extensor	0 (0%)	0 (0%)
Feet	1 (1.8%)	1 (1.8%)
Tibialis anterior	0 (0%)	0 (0%)
Posterior tibialis	1 (1.8%)	1 (1.8%)
Peroneus	0 (0%)	0 (0%)
	US enthesitis	PD positive
Feet	2 (3.5%)	2 (3.5%)
Achilles	2 (3.5%)	2 (3.5%)
Plantar Fascia	1 (1.8%)	0 (0%)

Table 2. US findings of patients with IA

Disclosure of Interests: Katherine López Gloria: None declared, Isabel Castrejon: None declared, Laura Trives Folguera Speakers bureau: ROCHE, Juan Carlos Nieto Speakers bureau: Pfizer, Abbvie, MSD, Novartis, Janssen, Lilly, Nordic Pharma, BMS, Gebro, FAES Farma, Roche, Sanofi, Belén Serrano Benavente: None declared, Julia Martínez-Barrio Consultant of: UCB Pharma, Javier Rivera: None declared, Carlos Gonzalez Consultant of: Gilead, Janssen, Novartis., Speakers bureau: Abbvie, Celgene, Gilead, Janssen, Novartis, Pfizer, Roche, Indalecio Monteagudo: None declared, Jose-Maria Alvaro-Gracia Grant/research support from: Abbvie, Elli-Lilly, MSD, Novartis, Pfizer, Consultant of: Abbvie, BMS, Janssen-Cilag, Elli-Lilly, MSD, Novartis, Pfizer, Sanofi, Tigenix, Roche, UCB, Paid instructor for: Elli-Lilly, Pfizer, Roche, Speakers bureau: Abbvie, BMS, Janssen-Cilag, Elli-Lilly, Gedeon Richter, MSD, Novartis, Pfizer, Sanofi, Tigenix, Roche, UCB, Juan Molina Collada: None declared

DOI: 10.1136/annrheumdis-2020-eular.6332

AB0206

CIRCULATING CENTROMERE PROTEIN F AUTOANTIBODIES FOR PREDICTING CLINICAL RESPONSE TO INFLIXIMAB IN RHEUMATOID ARTHRITIS

L. Lourido^{1,2}, C. Ruiz-Romero^{1,3}, F. Picchi¹, N. Diz-Rosales¹, S. Vilaboa-Galán¹, C. Fernández-López¹, J. A. Pinto Tasende¹, E. Perez-Pampin⁴, C. Regueiro Expósito⁴, A. Mera Varela⁴, A. Gonzalez⁴, K. Hambardzumyan⁵, S. Saevarsdottir⁵, P. Nilsson⁶, F. J. Blanco^{1,2}. ¹Grupo de Investigación de Reumatología (GIR), Instituto de Investigación Biomédica de A Coruña (INIBIC), Complejo Hospitalario Universitario de A Coruña (CHUAC), Sergas, Universidade da Coruña (UDC), As Xubias, ¹⁵⁰⁰⁶, A Coruña, Spain., A Coruña, Spain; ²RIER-RED de Inflamación y Enfermedades

Reumáticas, Spain, Madrid, Spain; ³CIBER-BBN Instituto de Salud Carlos III, Spain, Madrid, Spain; ⁴Laboratorio Investigación ¹⁰ and Rheumatology Unit, Instituto de Investigación Sanitaria-Hospital Clínico Universitario de Santiago, Santiago de Compostela, Spain, Santiago de Compostela, Spain; ⁵Unit of Rheumatology, Department of Medicine, Karolinska Institutet and Rheumatology Clinic, Karolinska University Hospital, Stockholm, Sweden, Stockholm, Sweden; ⁶Division of Affinity Proteomics, Department of Protein Science, KTH Royal Institute of Technology, SciLifeLab, Stockholm, Sweden, Stockholm, Sweden

Background: One third of patients with rheumatoid arthritis (RA) respond poorly to TNF inhibitors and related studies are inconsistent in predictive biomarkers. The identification of biomarkers that predict the treatment response prior to drug exposure is a current priority on the RA field. ACPA and RF are ubiquitously tested in RA patients, but other autoantibodies exist and may provide additional information on RA treatment response.

Objectives: This study aimed to identify circulating autoantibodies for predicting response to infliximab (IFX) in RA.

Methods: We profiled the autoantibody repertoire of baseline sera from 155 biologic naïve RA patients treated with IFX. The sera were provided by three independent clinical sources and distributed in one exploratory cohort (N=20) collected from Hospital Clínico Universitario de Santiago de Compostela (Spain), one replication cohort (N=61) collected from Hospital Universitario de A Coruña (Spain) and samples from the Swedish FarmacoTherapy (SWEFOT) trial (Sweden) (N=74) for clinical validation. The presence of autoantibodies and their levels in serum were analysed in association with EULAR clinical response at 6 months follow-up: good response (GR, N=56), moderate (MR, N=55) and non-response (NR, N=44). A suspension bead array platform built on protein fragments within Human Protein Atlas and selected from an initial untargeted screening using an array containing 42000 antigens was employed to identify the IgG and IgA autoantibodies on the exploratory cohort. A replication and validation phases were carried out on the other two serum sample cohorts. Meta-analysis and Receiver Operating Curves were performed in order to assess the clinical relevance of the findings observed.

Results: Meta-analysis revealed that the levels in serum of IgG autoantibodies against Centromere protein F (CENPF) are significantly increased in responders (good responders and moderate responders; N=111) to IFX compared to non-responders (N=44) (P=0.018). CENPF is a proliferation-associated and cell cycle-dependent centromere autoantigen that might be involved in the increased or abnormal cell proliferation that occurs during RA process. The combination of the anti-CENPF antibodies with clinical variables (age, sex, DAS28-ESR) resulted in the best model to discriminate the patients that will respond to IFX, showing an AUC of 0.756 (95% CI [0.639-0.874], P=0.001).

Conclusion: High serum levels of IgG anti-CENPF antibodies might be potentially useful to identify RA patients more likely to benefit from IFX

Disclosure of Interests: Lucía Lourido: None declared, Cristina Ruiz-Romero: None declared, flor picchi: None declared, Naomi Diz-Rosales: None declared, Sergio Vilaboa-Galán: None declared, Carlos Fernández-López: None declared, Jose Antonio Pinto Tasende: None declared, Eva Perez-Pampin: None declared, Cristina Regueiro Expósito: None declared, ANTONIO MERA VARELA: None declared, Antonio Gonzalez: None declared, Karen Hambardzumyan: None declared, Saedis Saevardottir Employee of: Part-time at deCODE Genetics/Amgen Inc, working on genetic research unrelated to this project, Peter Nilsson: None declared, Francisco J. Blanco Grant/research support from: Sanofi-Aventis, Lilly, Bristol MS, Amgen, Pfizer, Abbvie, TRB Chemica International, Glaxo SmithKline, Archigen Biotech Limited, Novartis, Nichi-iko pharmaceutical Co, Genentech, Janssen Research & Development, UCB Biopharma, Centrexion Therapeutics, Celgene, Roche, Regeneron Pharmaceuticals Inc, Biohope, Corbus Pharmaceutical, Tedec Meiji Pharma, Kiniksa Pharmaceuticals, Ltd, Gilead Sciences Inc, Consultant of: Lilly, Bristol MS, Pfizer

DOI: 10.1136/annrheumdis-2020-eular.4869

AB0207

CLINICAL IMPACT OF ANTI-CARP ANTIBODIES IN RHEUMATOID ARTHRITIS

M. Markovic¹, B. Glisic², M. Petronijevic². ¹Clinical Center of Montenegro, Clinic of Internal Medicine, Rheumatology Department, Podgorica, Montenegro; ²Military Medical Academy, Clinic of Rheumatology, Belgrade, Serbia

Background: Antibodies directed against carbamylated proteins (anti-CarP) have been recently introduced for the first time as a new biomarker in rheumatoid arthritis (RA) (1). Their presence is predictive for the development of RA (2). Anti-CarP antibodies are associated with the development of more severe forms of the disease in overall and anti-citrullinated peptide antibodies negative